INDIVIDUAL DIFFERENCES IN TEMPORAL FINE-STRUCTURE DETECTION ACROSS YOUNGER AND OLDER LISTENERS: DISENTANGLING SOURCES OF SENSORINERUAL HEARING LOSS USING A MODEL-BASED APPROACH

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ABSTRACT

Auditory modelling provides a powerful quantitative framework to study auditory mechanisms involved in psychoacoustic listening tasks. Here, we use a biophysically-inspired auditory model to simulate how different aspects of sensorineural hearing loss (SNHL) affect absolute thresholds in a monaural temporal fine structure (TFS) task, known as TFS1, and to which extent SNHL aspects can explain individual differences among listeners. We conducted the TFS1 test in 28 younger or older participants with normal and sloping high-frequency audiograms, respectively. Complex tones with statistically constant Hilbert envelopes but either regular or variable TFS had to be discriminated using a forced-choice paradigm to yield TFS thresholds expressed as Δf values in Hz. The observed threshold variability across participants ranged between 2.3 and 30.8 Hz. We compared our experimental findings to simulations that predicted an inter-subject Δf between 2.7 and 28.9 Hz for a combination of SNHL profiles. The simulations were obtained for a constant-stimulus version of the experiment and required a constant Hilbert envelope within each trial, otherwise the simulations were dominated by the envelope differences between the two intervals in the trial. Under this simulation constraint, our results suggest that synaptopathy might underlie individual differences among listeners with normal audiograms, and that it also plays a bigger role than audibility when both deficits are present.

1. INTRODUCTION

Reduced speech intelligibility in noisy listening scenarios does not only affect listeners with diagnosed sensorineural hearing loss (SNHL), but is also a common complaint among people who have normal-hearing audiograms [1]. Previous studies have in fact shown that hearing sensitivity as measured using audiograms can be poorly related with performance in experiments of speech intelligibility [2]. The listener's performance in psychoacoustic tasks with non-speech sounds, including tests targeting temporal fine-structure (TFS) and temporal envelope, is a better predictor of speech intelligibility problems [2–4]. In this extended abstract, we focus on a monaural TFS task, known as TFS1 [5], that was found to be significantly correlated with reduced speech-in-noise intelligibility in a previous study of our group [6]. The experimental results are compared with simulations of the same TFS1 task using a biophysically-inspired computational model of the human auditory periphery and brainstem [7, 8]. The simulations consider two forms of SNHL: Hair-cell damage and synaptopathy. Hair-cell damage is simulated using cochlear gain profiles matched to average normal and impaired audiograms, and synaptopathy is simulated as a gradual removal of auditory nerve (AN) synapses, ranging from a healthy AN population (no synaptopathy) to 50% AN survival. We hypothesised that if normal audiograms do not necessarily reflect problems in temporal TFS coding (as is the case for speech intelligibility in noise), then synaptopathy is expected to affect the simulated TFS1 thresholds more drastically than outer-hair-cell damage.

2. METHODS AND RESULTS

Experiment: The experiment was a monaural temporal-fine-structure task (TFS1) which has been described by Moore and Sek [5] and implemented in the TFS software

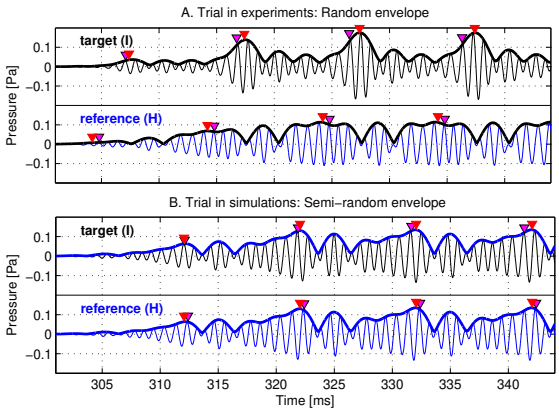

Figure 1. Second burst of one target (inharmonic, I) and reference (harmonic, H) stimulus as used in the experiments (panel **A**) and in the simulations (panel **B**). For H bursts both the TFS and Hilbert envelope have constant relative phases (see the position of the pink and red arrows in **A**–**B**, bottom), while for I bursts the TFS flows more quickly (repetition rate of f_0+Δf) than the envelope (rate of f_0), see the relative arrow positions in **A**–**B**, top.

[9]. Participants were asked to listen, using HD200 headphones, to two trains of four 200-ms tone bursts presented at 70 dB SPL and identify which of the two trains contained bursts with changing pitch (the target). An example of target sound (harmonic-inharmonic HIHI bursts) and reference sound (harmonic HHHH bursts) used in such 2-AFC paradigm are shown in Fig. 1**A**. The pitch-changing HIHI burst had subsequent harmonic (f_0=100 Hz) and inharmonic tones ($f_0+\Delta f$), using partials 9 to 13, where Δf was the tracking variable (higher Δf was easier to detect). The Δf thresholds for the younger participants (yNH, N=15, 24.5y$\pm$2.3, normal-hearing audiograms) were between 2.3 and 15.8 Hz and for the older group (oHI, N=13, 65.2y$\pm$1.8; flat audiograms of 10 dB HL up to 1 kHz, sloping-audiogram thereafter with 45 dB HL at 8 kHz) were between 3.6 and 30.8 Hz (grey circles in Fig. 2**A**).

Simulations: The same task was simulated using the auditory model of Verhulst et al. [7] v1.2 [8] where fixed Δf values (1, 5, 25, 50 Hz) were tested using a fixed Hilbert envelope in target and reference sounds, as shown in Fig. 1**B**. The TFS1 thresholds were derived using the methods outlined in [10], assuming that detection-cue curves of percentiles 25, 50, and 75 (dashed and continuous lines, Fig. 2**B**) are related to the interquartile range and median of the simulated TFS1 thresholds. This approach resulted in simulated Δf thresholds (Fig. 2**A**) whose variability increased with increasing synaptopathy: For the NH audiogram profile (Flat00, in blue) the 2.8-8 Hz Δf range increased up to 3.1-13.6 Hz with synaptopathy (7-0-0). For the HI profile (Flat10-Slope45, in red) the Δf range increased from 2.7-13.6 Hz (13-3-3) to 2.8-28.9 Hz (7-0-0).

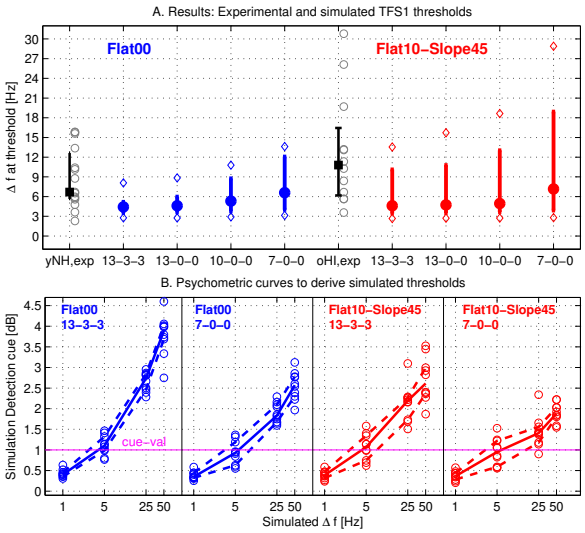

Figure 2. **A**. Experimental (black) and simulated TFS1 thresholds for NH (Flat00, in blue) and HI audiograms (Flat10-Slope45, in red) for different degrees of synaptopathy (see text). The error bars indicate interquartile ranges, and diamond markers the minimum and maximum simulated thresholds. **B**. The simulated thresholds were obtained from the intersection between the detection cue curves and a detection cue value (det_val=1 dB, pink line). Four sets of curves are shown: Flat00 and Flat10-Slope45, with no synaptopathy and 50% AN loss.

3. DISCUSSION AND FURTHER WORK

The experimental data for younger yNH and older oHI participants showed a high inter-subject performance variability that could, to a great extent, be explained by the simulated variance caused by SNHL. Synaptopathy played a more relevant role in explaining this simulated variability than hair cell loss. The interaction of both forms of SNHL (HI audiogram and synaptopathy) resulted in a worse performance than the use of synaptopathy profiles alone, with more than a doubling of the Δf thresholds, particularly visible in thresholds related to percentiles 99 and 75. During the presentation we will address which aspects of the auditory modelling were crucial and had an influence in our results. We will also provide an in-depth discussion on the experimental outcomes and model simulations.

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4. REFERENCES

[1] S. Anderson, S. Gordon-Salant, and J. Dubno, “Hearing and aging effects on speech understanding: Challenges and solutions,” *Acoust. Today*, vol. 14, no. 4, pp. 10–18, 2018.

[2] A. Papakonstantinou, O. Strelcyk, and T. Dau, “Relations between perceptual measures of temporal processing, auditory-evoked brainstem responses and speech intelligibility in noise,” *Hear. Res.*, vol. 280, pp. 30–7, 2011.

[3] C. Lorenzi, G. Gilbert, H. Carn, S. Garnier, and B. Moore, “Speech perception problems of the hearing impaired reflect inability to use temporal fine structure,” *Proc. Nat. Ac. Sci.*, vol. 103, pp. 18866–9, 2006.

[4] K. Hopkins, B. Moore, and M. Stone, “Effects of moderate cochlear hearing loss on the ability to benefit from temporal fine structure information in speech,” *J. Acoust. Soc. Am.*, vol. 123, pp. 1140–53, 2008.

[5] B. Moore and A. Sek, “Development of a fast method for determining sensitivity to temporal fine structure,” *Int. J. Audiol.*, vol. 48, pp. 161–71, 2009.

[6] M. Garrett, V. Vasilkov, M. Mauermann, and S. Verhulst, “Objective and behavioral markers of low- and high-frequency processing and their contribution to speech intelligibility in healthy and impaired ears,” in *43rd ARO Midwinter Meeting*, p. 165, 2020.

[7] S. Verhulst, A. Altoè, and V. Vasilkov, “Computational modeling of the human auditory periphery: Auditory-nerve responses, evoked potentials and hearing loss,” *Hear. Res.*, vol. 360, pp. 55–75, 2018.

[8] A. Osses Vecchi and S. Verhulst, “Release note on version 1.2 of the Verhulst et al. 2018 model of the human auditory system: Calibration and reference simulations,” *ArXiv*. ArXiv:1912.10026.

[9] A. Sek and B. Moore, “Implementation of two tests for measuring sensitivity to temporal fine structure,” *Int. J. Audiol.*, vol. 51, pp. 58–63, 2012.

[10] A. Osses Vecchi, F. Ernst, and S. Verhulst, “Performance variability in supra-threshold detection tasks due to stimulus properties and sensorineural hearing loss,” (in preparation).